Diagnosis of Past Fish and Wildlife Population Declines Supports Establishment of Water Quality Criteria to Prevent Future Dioxin Toxicity Events

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The lake trout, a native keystone predator fish species, was extirpated in Lake Ontario around 1960. Population data for 120 years, including the last 30 years during which slow progress toward sustainable natural reproduction by stocked trout was observed, provided a unique opportunity to evaluate the applicability of exposure and toxicity hazard prediction models. These models are needed for derivation of chemical residue-based water quality criteria and site-specific risk assessment methods that can protect populations of aquatic species and wildlife that feed on them. Analyses of radionuclide-dated, 1-cm sections of sediment cores indicated that Lake Ontario received large loadings of polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and biphenyls (PCBs) during the period of 1940-1970. Slowly declining concentrations in sediments and fish indicate that loadings were dramatically reduced after 1970. Lake trout were determined to be very sensitive to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) during early life-stage (ELS) development. The key question was whether the lake trout population decline, extirpation, and subsequent lack of natural reproduction in reintroduced trout could be attributable to ELS toxicity. Measurements of concentrations of PCDDs, PCDFs, and PCBs with known toxicity like TCDD were available for lake trout only back to 1978. Therefore, reconstruction of the contribution made by ELS toxicity to the decline of lake trout that started in the 1930s depended on exposure predictions made from concentrations of each chemical with depth in the sediment cores. Lake trout embryo-specific biota sediment accumulation factors (BSAFs), adjusted for temporal changes in relative contaminant distributions between water and sediments, transformed concentrations in sediments over time to concentrations in eggs. The dioxin additive toxicity equivalence model was used to calculate the net ELS toxicity associated with the predicted mixture of dioxin-like chemicals in lake trout eggs. By 1940, the predicted sac fry mortality alone explains the decline and subsequent loss of lake trout. Multiple lines of evidence were used to validate the predicted exposures and consequent degrees of ELS mortality over time. Uncertainties for consequences of post-1985 exposures, which were below a threshold for overt mortality, highlight areas for additional research. Although less thoroughly analyzed and specific for dioxin toxicity, population declines of other Lake Ontario fish and wildlife species with different sensitivities appear to be similarly associated with high concentrations of persistent bioaccumulative toxic chemicals in their tissues or diet.